WEST Search History

DATE: Tuesday, July 30, 2002

Set Name side by side	Query	Hit Count	Set Name result set
	DB=USPT,PGPB; PLUR=YES; OP=ADJ		
L7	l1 and (cd63 same (reduc$ or inhibi$))	10	L7
L6	l1 and (cd63 same (reduc$ or inhibi$))	9	L6
L5	l1 and (cd63 near5 (reduc$ or inhibi$))	0	L5
L4	L3 and ((inhib$ or reduc$) near5 l1)	8	L4
L3	l1 and L2	35	L3
L2	cd63	112	L2
L1	hiv or (human immunodef$)	18792	L1

END OF SEARCH HISTORY

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FILE 'HOME' ENTERED AT 12:28:49 ON 30 JUL 2002

=> FIL BIOSIS MEDLINE SCISEARCH CA
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILE 'BIOSIS' ENTERED AT 12:28:57 ON 30 JUL 2002
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FILE 'MEDLINE' ENTERED AT 12:28:57 ON 30 JUL 2002

FILE 'SCISEARCH' ENTERED AT 12:28:57 ON 30 JUL 2002
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FILE 'CA' ENTERED AT 12:28:57 ON 30 JUL 2002
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=> s cd63
L1         1690 CD63

=> s hiv or (human immunodef?)
L2       403777 HIV OR (HUMAN IMMUNODEF?)

=> s l1 and l2
L3           26 L1 AND L2

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4           11 DUP REM L3 (15 DUPLICATES REMOVED)

=> s l4 and py=<2000
   1 FILES SEARCHED...
L5           10 L4 AND PY=<2000

=> d l5 1-10 ibib abs

L5   ANSWER 1 OF 10  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER:    2000:384795  BIOSIS
DOCUMENT NUMBER:     PREV200000384795
TITLE:               Hypericin inactivates viruses in platelet concentrates.
AUTHOR(S):           Seifried, E. (1); Mueller, M. (1); Willkommen, H.;
                     Scheiblauer, H.; Norley, S.; Kirchmaier, C. M. (1)
CORPORATE SOURCE:    (1) RC Blood Donor Service Center, Inst. Transfusion
                     Medicine/Immunohaematology, Frankfurt Germany
SOURCE:              Vox Sanguinis, (July, 2000) Vol. 78, No. Suppl.
                     1, pp. 0104. print.
                     Meeting Info.: 26th Congress of the International Society
                     of Blood Transfusion Vienna, Austria July 09-14, 2000
                     International Society of Blood Transfusion
                     . ISSN: 0042-9007.
DOCUMENT TYPE:       Conference
LANGUAGE:            English
SUMMARY LANGUAGE:    English

L5   ANSWER 2 OF 10  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER:    1999:167004  BIOSIS
DOCUMENT NUMBER:     PREV199900167004
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TITLE: Regulation of class II production after **HIV**-1 infection.
AUTHOR(S): Kraus, T.; Chen, H.; Becker, K.; Rakoff, K. S.; Sperber, K.
CORPORATE SOURCE: Mt. Sinai Sch. Med., New York, NY 10029 USA
SOURCE: FASEB Journal, (**March 12, 1999**) Vol. 13, No. 4 PART 1, pp. A292.
Meeting Info.: Annual Meeting of the Professional Research Scientists for Experimental Biology 99 Washington, D.C., USA April 17-21, 1999
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English

L5 ANSWER 3 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:116428 BIOSIS
DOCUMENT NUMBER: PREV199800116428
TITLE: Enhanced activation of platelets with abnormal release on RANTES in **human immunodeficiency** virus type 1 infection.
AUTHOR(S): Holme, Pal Andre; Muller, Fredrik; Solum, Nils Olav; Brosstad, Frank; Froland, Stig S.; Aukrust, Pal (1)
CORPORATE SOURCE: (1) Section Clinical Immunol. Infectious Diseases, Med. Dep. A, Rikshospitalet, N-0027 Oslo Norway
SOURCE: FASEB Journal, (**Jan., 1998**) Vol. 12, No. 1, pp. 79-90.
ISSN: 0892-6638.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Besides their role in hemostasis, platelets are involved in inflammatory and immunological processes, and we hypothezise that platelet activation may play an immunopathogenetic role in **HIV**-1 infection. Blood was drawn from 15 controls and 20 **HIV**-1-infected patients with normal platelet counts, classified into groups of non-AIDS and AIDS. Platelet activation was detected using flow cytometry with mAbs against the release markers P-selectin and **CD63**, mAb against GPIb, and the probe annexin V detecting surface exposure of aminophospholipids. The amount of microvesicles was measured using mAb against GPIIIa. Compared to controls, blood samples from **HIV**-1-infected patients showed significantly enhanced levels of microvesicles and activated platelets as detected by their exposure of P-selectin, **CD63**, and aminophospholipids, as well as reduction in GPIb expression. Increased expression of P-selectin and amounts of microvesicles were most pronounced in advanced clinical and immunological disease. When studying the effect of **HIV**-1 protease inhibitor therapy (indinavir) on platelet activation, we found that concomitant with a profound decrease in plasma viral load, there was a near normalization of several of the parameters reflecting enhanced platelet activation. Finally, we demonstrated that platelets may be an important source of the chemokine RANTES in **HIV**-1-infected patients. Although both unstimulated and SFLLRN-stimulated platelets from asymptomatic patients had enhanced release of RANTES, platelets from AIDS patients were characterized by markedly enhanced spontaneous, but decreased SFLLRN-stimulated release of this chemokine. Taken together, these results, which demonstrate for the first time increased platelet activation in **HIV**-1-infected patients with normal platelet counts, may represent a previously unrecognized immunopathogenic factor in **HIV**-1 infection.

L5 ANSWER 4 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1997:213378 BIOSIS
DOCUMENT NUMBER: PREV199799519882
TITLE: Cell membrane vesicles are a major contaminant of gradient-enriched **human immunodeficiency**

virus type-1 preparations.

AUTHOR(S): Gluschankof, Pablo (1); Mondor, Isabelle; Gelderblom, Hans R.; Sattentau, Quentin J.

CORPORATE SOURCE: (1) Centre Immunol. Marseille-Luminy, Case 906, 13288 Marseille France

SOURCE: Virology, (1997) Vol. 230, No. 1, pp. 125-133.
ISSN: 0042-6822.

DOCUMENT TYPE: Article

LANGUAGE: English

AB During preliminary experiments to establish the proportion of virus-coded p24 protein to virus membrane-associated HLA-DR in gradient-enriched **HIV**-1 preparations, we became aware of a large variability between experiments. In order to determine whether HLA-DR-containing cellular material was contaminating the virus preparations, we carried out enrichment by gradient centrifugation of clarified supernatants from noninfected cells and tested this material for HLA-DR content. We found that, independently of the cell type used, gradient enrichment resulted in the isolation of large quantities of HLA-DR containing material which banded at a density overlapping that of infectious **HIV**. Electron microscopy of gradient-enriched preparations from supernatants of virus-infected cells revealed an excess of vesicles with a size range of about 50-500 nm, as opposed to a minor population of virus particles of about 100 nm. Electron micrographs of infected cells showed polarized vesiculation of the cell membrane, and virus budding was frequently colocalized with nonviral membrane vesiculation. Analysis of the cellular molecules present in the fractions containing virus or exclusively cellular material demonstrated that virus and cellular vesicles share several cellular antigens, with the exception of CD43 and **CD63**, found mainly at the virus surface, and HLA-DQ, which was found only in the cellular vesicles.

L5 ANSWER 5 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 1994:78168 BIOSIS

DOCUMENT NUMBER: PREV199497091168

TITLE: Association of host cell surface adhesion receptors and other membrane proteins with **HIV** and SIV.

AUTHOR(S): Orentas, Rimas J.; Hildreth, James E. K. (1)

CORPORATE SOURCE: (1) Leukocyte Immunochem. Lab., Johns Hopkins Univ. Sch. Med., Dep. Pharmacol. and Molecular Sci., 725 N. Wolfe St., Baltimore, MD 21205 USA

SOURCE: AIDS Research and Human Retroviruses, (1993) Vol. 9, No. 11, pp. 1157-1165.
ISSN: 0889-2229.

DOCUMENT TYPE: Article

LANGUAGE: English

AB We have developed a MAb-based capture assay to study the association of host cell membrane proteins with **HIV** and SIV. Class I and II MHC proteins were found to be associated with **HIV** as previously described. In addition to these molecules a number of other host molecules were found to be acquired by **HIV**, including CD71, **CD63** , CD43, and CD8. We have demonstrated that the major leukocyte adhesion receptors LFA-1 (CD11A/CD18) and CD44 are also associated with **HIV** . The level of surface expression of host membrane proteins did not predict relative expression (capture efficiency) of the virus. The use of virus-susceptible indicator cells showed that the assay involved host membrane protein-mediated capture of infectious **HIV** and SIV particles. Our data indicate that **HIV** and SIV acquire a number of host membrane proteins including adhesion receptors and that this process may be nonrandom. The acquisition of host cell adhesion receptors by **HIV** and SIV could have profound effects on the biology of the viruses, including binding, infectivity, and tropism.

L5 ANSWER 6 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1993:171318 BIOSIS
DOCUMENT NUMBER: PREV199395092368
TITLE: Host cell membrane proteins on **human immunodeficiency** virus type 1 after in vitro infection of H9 cells and blood mononuclear cells: An immuno-electron microscopic study.
AUTHOR(S): Meerloo, Timo (1); Sheikh, Mubasher A. (1); Bloem, Andries C.; De Ronde, Anthony; Schutten, Martin; Van Els, Cecile A. C.; Roholl, Paul J. M.; Joling, Piet (1); Goudsmit, Jaap; Schuurman, Henk-Jan
CORPORATE SOURCE: (1) Div. Histochem. Electron Microscopy, Dep. Pathol. Internal Med., University Hospital, PO Box 85.500, 3508 GA Utrecht Netherlands Antilles
SOURCE: Journal of General Virology, (1993) Vol. 74, No. 1, pp. 129-135.
ISSN: 0022-1317.
DOCUMENT TYPE: Article
LANGUAGE: English
AB **Human immunodeficiency** virus type 1 (**HIV**-1)-infected H9 and blood mononuclear cells (MNCs) were studied by immunogold electron microscopy for the presence of **HIV**-1 gag p24 protein, env gp41 and gp120 proteins, and host cell molecules CD4, CD11a, CD25, CD54, **CD63**, HLA class I and HLA-DR. Uninfected H9 cells and MNC membranes labelled for CD4, HLA class I and class II, and, at low density, CD11a and CD54; lysosomal structures in the cytoplasm labelled for **CD63**. The infected cell surface showed immunolabelling for **HIV**-1 proteins, as did budding particle-like structures. Immunogold labelling of the cell membrane for CD4 was almost non-existent. The level of immunolabelling for CD11a and CD54 on infected cells was greater than that on uninfected cells; this is presumably related to a state of activation during virus synthesis. Budding particle-like structures and free virions in the intercellular space were immunogold-labelled for all host cell markers investigated. This was confirmed by double immunogold labelling using combination of **HIV**-1 gag p24 labelling and labelling for the respective host cell molecule. We conclude that virions generated in **HIV**-1-infected cells concentrate host-derived molecules on their envelope. Also molecules with a prime function in cellular adhesion concentrate on the virion.

L5 ANSWER 7 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1993:34898 BIOSIS
DOCUMENT NUMBER: PREV199395023098
TITLE: Modulation of cell surface molecules during **HIV**-1 infection of H9 cells: An immunoelectron microscopic study.
AUTHOR(S): Meerloo, Timo; Parmentier, Henk K.; Osterhaus, Albert D. M. E.; Goudsmit, Jaap; Schuurman, Henk-Jan (1)
CORPORATE SOURCE: (1) Div. Histochemistry, Electron Microscopy, Dep. Pathology, Univ. Hosp., P.O. Box 85.500, 3508 GA Utrecht Netherlands Antilles
SOURCE: AIDS (Philadelphia), (1992) Vol. 6, No. 10, pp. 1105-1116.
ISSN: 0269-9370.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Objective: To study cell surface molecules and **HIV**-1 proteins on H9 cells 2 days after infection by immunogold electron microscopy, either in single or in double labelling using combinations of host cell-derived molecules and **HIV**-1 proteins. Design and methods: The presence of host cell antigens CD3, CD4 and human leukocyte antigen-DR (HLA-DR) and **HIV**-1 antigens gag p15, p17, p24 and env gp41 was evaluated using immunocytochemistry at the light microscopic level. H9 cells 2 days after infection were processed for conventional transmission electron microscopy

and cryo-ultramicrotomy. Leukocyte antigens investigated were CD2, CD3, CD4 (two antibodies), CD5, CD8, CD25, CD30, **CD63** antigens and HLA-DR; **HIV**-1-encoded antigens were gag p24, pol reverse transcriptase, and env gp41 and gp120. Double immunogold labelling was performed using reagents with different sized gold particles. For leukocyte markers, the labelling density of the cell membrane was assessed quantitatively on uninfected and infected H9 cells. Results: Infected cells revealed the presence of gag p24, pol, and env gp41 and gp120 antigens on **HIV**-1 virions. Uninfected H9 cells showed a random distribution of cell surface molecules, including CD4 antigen, along the plasma membrane. The **CD63** antigen, a lysosomal membrane glycoprotein, was located mainly in the cytoplasm of uninfected cell. Cells 2 days after infection showed CD4 labelling on sites where virions were budding from or attached to the cell surface and on free virions. Virions also showed labelling by CD3, CD5, CD25, CD30 and **CD63** antibodies and anti-HLA-DR. Compared with uninfected cells, a significantly lower density was found on infected cells in labeling for CD4, CD5, and anti-HLA-DR. A significantly higher density on cells 2 days after infection was seen in **CD63** labelling. Conclusions: During the first phase of infection host cell molecules concentrate on budding structures and newly generated **HIV**-1 virions. This phenomenon might contribute to the disappearance of these molecules (like the CD4 molecule) from the cell membrane and infection.

L5 ANSWER 8 OF 10 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 133:340273 CA
TITLE: Methods and formulations for targeting infectious agents bearing host cell proteins
INVENTOR(S): Bergeron, Michel G.; Desormeaux, Andre; Tremblay, Michel J.
PATENT ASSIGNEE(S): Infectio Recherche Inc., Can.
SOURCE: PCT Int. Appl., 45 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000066173	A2	20001109	WO 2000-CA469	20000503 <--
WO 2000066173	A3	20010809		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1173220	A2	20020123	EP 2000-922374	20000503
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PRIORITY APPLN. INFO.: CA 1999-2270600 A 19990503
WO 2000-CA469 W 20000503

AB A formulation is disclosed for the treatment of diseases caused by an infectious agent which acquires host membranes protein during its life cycle. The formulation is a targeting pharmaceutical compn. It comprises a ligand capable of binding the host membrane proteins coupled to a lipid-comprising vesicle, which may comprise or not a drug effective in the treatment of the disease. Specific liposomes bearing anti-HLA-DR or

anti-CD4 antibodies comprising or not antiviral drugs, namely anti-**HIV** drugs, are disclosed and claimed. A method of formulation as well as a method of using the formulation in the treatment of a disease are also disclosed.

L5 ANSWER 9 OF 10 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 130:264436 CA
TITLE: Methods of replicating virus in monocyte-derived macrophage cultures
INVENTOR(S): Soderberg-naucler, Cecilia; Fish, Kenneth N.; Moses, Ashlee; Streblow, Daniel; Nelson, Jay
PATENT ASSIGNEE(S): Oregon Health Sciences University, USA
SOURCE: PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9916891	A1	19990408	WO 1998-US20749	19980930 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
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AU 9895993	A1	19990423	AU 1998-95993	19980930 <--
AU 738685	B2	20010927		
EP 1023451	A1	20000802	EP 1998-949728	19980930 <--
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US 6225048	B1	20010501	US 1998-164221	19980930
JP 2001518306	T2	20011016	JP 2000-513960	19980930
US 2001055755	A1	20011227	US 2001-810328	20010315

PRIORITY APPLN. INFO.: US 1997-60583P P 19971001
US 1998-164221 A1 19980930
WO 1998-US20749 W 19980930

AB The present invention provides methods of latent virus reactivation in monocyte-drived macrophages through allogeneic stimulation of peripheral blood mononuclear cells (PBMC), methods of culturing virus, and cultures of virally permissive monocyte-derived macrophages. To det. whether cytokines or other sol. factors are sufficient to differentiate monocytes to human cytomegalovirus-permissive monocyte-derived macrophages (MDM), allogeneically stimulated MDM conditioned culture medium was used to differentiate CD14+ monocytes obtained from naturally infected seropos. donors. A transwell system was used to sep. the monocytes from a single seropos. donor from an allo-reaction of two seroneg. donors. Conditioned medium was sufficient to differentiate monocytes into MDM with a similar morphol. and viral permissiveness as the parallel allo-MDM cell cultures.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 10 OF 10 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 129:92575 CA
TITLE: Method for characterization of abnormal cells using multiple antibody- or ligand-coated particles
INVENTOR(S): Fodstad, Oystein; Hoifodt, Hanne Kleppe

PATENT ASSIGNEE(S): Norway
SOURCE: PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9828622	A1	19980702	WO 1997-NO342	19971216 <--
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RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
NO 9605531	A	19980622	NO 1996-5531	19961220 <--
AU 9878752	A1	19980717	AU 1998-78752	19971216 <--
AU 728190	B2	20010104		
EP 951645	A1	19991027	EP 1997-949270	19971216 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: NO 1996-5531 A 19961220
WO 1997-NO342 W 19971216

AB A method to detect and phenotype target cells in cell suspensions uses particles coated with antibodies/ligands directed to antigenic determinants/receptors expressed on the target cells. The method is characterized in that several types of particles are used and each type of particle is instrumentally or visually separable by fluorescence, color and size. Each type of particle is coated with a different antibody or ligand. The particles are incubated simultaneously or sequentially with cell suspensions contg. the target cells, in connection or not with a per se known enrichment procedure. A kit using the method is also disclosed. A suspension of ascitic cells was incubated with different antibody-coated fluorescent particles and paramagnetic immunobeads. The cells were detd. to be malignant and epithelial in nature based on the antibody particles that bound to the cells.

=> s o'brien, W?/au; s (Ferbas or Grovit-ferbas), K?/au
MISMATCHED QUOTE 'O'BRIEN,'
Quotation marks (or apostrophes) must be used in pairs,
one before and one after the expression you are setting
off or masking.

=> s obrien, W?/au; s (Ferbas or Grovit-ferbas), K?/au
L6 1136 OBRIEN, W?/AU

MISSING OPERATOR IT-FERBAS), K?/AU
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s obrien, W?/au; s (Ferbas, K?/au) or (Grovit-ferbas, K?/au)
L7 1136 OBRIEN, W?/AU

L8 26 (FERBAS, K?/AU) OR (GROVIT-FERBAS, K?/AU)

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=> s (l7 or l8) and cd63
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=> s (l7 or l8) and hiv
L10          73 (L7 OR L8) AND HIV

=> d his

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     FILE 'BIOSIS, MEDLINE, SCISEARCH, CA' ENTERED AT 12:28:57 ON 30 JUL 2002
L1          1690 S CD63
L2        403777 S HIV OR (HUMAN IMMUNODEF?)
L3            26 S L1 AND L2
L4            11 DUP REM L3 (15 DUPLICATES REMOVED)
L5            10 S L4 AND PY=<2000
L6          1136 S OBRIEN, W?/AU
L7          1136 S OBRIEN, W?/AU
L8            26 S (FERBAS, K?/AU) OR (GROVIT-FERBAS, K?/AU)
L9             0 S (L7 OR L8) AND CD63
L10           73 S (L7 OR L8) AND HIV
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=> FIL BIOSIS MEDLINE SCISEARCH CA
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                   ENTRY    SESSION
FULL ESTIMATED COST                                 0.42       0.42

FILE 'BIOSIS' ENTERED AT 14:59:28 ON 30 JUL 2002
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FILE 'MEDLINE' ENTERED AT 14:59:28 ON 30 JUL 2002

FILE 'SCISEARCH' ENTERED AT 14:59:28 ON 30 JUL 2002
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=> s cd63
L1         1690 CD63

=> lentivir?
LENTIVIR? IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s lentivir?
L2        59597 LENTIVIR?

=> s l1 and l2
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L6           11 DUP REM L5 (1 DUPLICATE REMOVED)

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L7           10 L6 AND PY<=2000

=> d l7 1-10 ibib abs

L7   ANSWER 1 OF 10  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER:    2000:384795  BIOSIS
DOCUMENT NUMBER:     PREV200000384795
TITLE:               Hypericin inactivates viruses in platelet concentrates.
AUTHOR(S):           Seifried, E. (1); Mueller, M. (1); Willkommen, H.;
                     Scheiblauer, H.; Norley, S.; Kirchmaier, C. M. (1)
CORPORATE SOURCE:    (1) RC Blood Donor Service Center, Inst. Transfusion
                     Medicine/Immunohaematology, Frankfurt Germany
SOURCE:              Vox Sanguinis, (July, 2000) Vol. 78, No. Suppl.
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1, pp. 0104. print.
Meeting Info.: 26th Congress of the International Society of Blood Transfusion Vienna, Austria July 09-14, 2000 International Society of Blood Transfusion
. ISSN: 0042-9007.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L7 ANSWER 2 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1999:167004 BIOSIS
DOCUMENT NUMBER: PREV199900167004
TITLE: Regulation of class II production after HIV-1 infection.
AUTHOR(S): Kraus, T.; Chen, H.; Becker, K.; Rakoff, K. S.; Sperber, K.
CORPORATE SOURCE: Mt. Sinai Sch. Med., New York, NY 10029 USA
SOURCE: FASEB Journal, (**March 12, 1999**) Vol. 13, No. 4 PART 1, pp. A292.
Meeting Info.: Annual Meeting of the Professional Research Scientists for Experimental Biology 99 Washington, D.C., USA April 17-21, 1999
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English

L7 ANSWER 3 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:116428 BIOSIS
DOCUMENT NUMBER: PREV199800116428
TITLE: Enhanced activation of platelets with abnormal release on RANTES in human immunodeficiency virus type 1 infection.
AUTHOR(S): Holme, Pal Andre; Muller, Fredrik; Solum, Nils Olav; Brosstad, Frank; Froland, Stig S.; Aukrust, Pal (1)
CORPORATE SOURCE: (1) Section Clinical Immunol. Infectious Diseases, Med. Dep. A, Rikshospitalet, N-0027 Oslo Norway
SOURCE: FASEB Journal, (**Jan., 1998**) Vol. 12, No. 1, pp. 79-90.
ISSN: 0892-6638.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Besides their role in hemostasis, platelets are involved in inflammatory and immunological processes, and we hypothezise that platelet activation may play an immunopathogenetic role in HIV-1 infection. Blood was drawn from 15 controls and 20 HIV-1-infected patients with normal platelet counts, classified into groups of non-AIDS and AIDS. Platelet activation was detected using flow cytometry with mAbs against the release markers P-selectin and **CD63**, mAb against GPIb, and the probe annexin V detecting surface exposure of aminophospholipids. The amount of microvesicles was measured using mAb against GPIIIa. Compared to controls, blood samples from HIV-1-infected patients showed significantly enhanced levels of microvesicles and activated platelets as detected by their exposure of P-selectin, **CD63**, and aminophospholipids, as well as reduction in GPIb expression. Increased expression of P-selectin and amounts of microvesicles were most pronounced in advanced clinical and immunological disease. When studying the effect of HIV-1 protease inhibitor therapy (indinavir) on platelet activation, we found that concomitant with a profound decrease in plasma viral load, there was a near normalization of several of the parameters reflecting enhanced platelet activation. Finally, we demonstrated that platelets may be an important source of the chemokine RANTES in HIV-1-infected patients. Although both unstimulated and SFLLRN-stimulated platelets from asymptomatic patients had enhanced release of RANTES, platelets from AIDS patients were characterized by markedly enhanced spontaneous, but decreased SFLLRN-stimulated release of this chemokine. Taken together,

these results, which demonstrate for the first time increased platelet activation in HIV-1-infected patients with normal platelet counts, may represent a previously unrecognized immunopathogenic factor in HIV-1 infection.

L7 ANSWER 4 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1997:213378 BIOSIS
DOCUMENT NUMBER: PREV199799519882
TITLE: Cell membrane vesicles are a major contaminant of gradient-enriched human immunodeficiency virus type-1 preparations.
AUTHOR(S): Gluschankof, Pablo (1); Mondor, Isabelle; Gelderblom, Hans R.; Sattentau, Quentin J.
CORPORATE SOURCE: (1) Centre Immunol. Marseille-Luminy, Case 906, 13288 Marseille France
SOURCE: Virology, (1997) Vol. 230, No. 1, pp. 125-133. ISSN: 0042-6822.
DOCUMENT TYPE: Article
LANGUAGE: English
AB During preliminary experiments to establish the proportion of virus-coded p24 protein to virus membrane-associated HLA-DR in gradient-enriched HIV-1 preparations, we became aware of a large variability between experiments. In order to determine whether HLA-DR-containing cellular material was contaminating the virus preparations, we carried out enrichment by gradient centrifugation of clarified supernatants from noninfected cells and tested this material for HLA-DR content. We found that, independently of the cell type used, gradient enrichment resulted in the isolation of large quantities of HLA-DR containing material which banded at a density overlapping that of infectious HIV. Electron microscopy of gradient-enriched preparations from supernatants of virus-infected cells revealed an excess of vesicles with a size range of about 50-500 nm, as opposed to a minor population of virus particles of about 100 nm. Electron micrographs of infected cells showed polarized vesiculation of the cell membrane, and virus budding was frequently colocalized with nonviral membrane vesiculation. Analysis of the cellular molecules present in the fractions containing virus or exclusively cellular material demonstrated that virus and cellular vesicles share several cellular antigens, with the exception of CD43 and **CD63**, found mainly at the virus surface, and HLA-DQ, which was found only in the cellular vesicles.

L7 ANSWER 5 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1995:298908 BIOSIS
DOCUMENT NUMBER: PREV199598313208
TITLE: Infection with Human T-Lymphotropic Virus Types I and II Results in Alterations of Cellular Receptors, Including the Up-Modulation of T-Cell Counterreceptors CD40, CD54, and CD80 (B7-1.
AUTHOR(S): Dezzutti, S. Charlene (1); Rudolph, Donna L.; Lal, Renu B.
CORPORATE SOURCE: (1) Retrovirus Diseases Branch, Centers Disease Control Prevention, 1600 Clifton Rd., MS G19, Atlanta, GA 30333 USA
SOURCE: Clinical and Diagnostic Laboratory Immunology, (1995) Vol. 2, No. 3, pp. 349-355. ISSN: 1071-412X.
DOCUMENT TYPE: Article
LANGUAGE: English
AB To examine the phenotypic alterations associated with human T-lymphotropic virus types I and II (HTLV-I and -II) infection, long-term cell lines (n = 12 HTLV-I cell lines; n = 11 HTLV-II cell lines; n = 6 virus-negative cell lines) were analyzed for the cell surface expression of various lineage markers (i.e., myeloid, progenitor, and leukocyte), integrin receptors, and receptor-counterreceptor (R-CR) pairs responsible for cellular activation. As expected, all cell lines expressed the markers

characterizing the leukocyte lineage (CD43, CD44, and CD53). Of the progenitor-myeloid markers examined (CD9, CD13, CD33, CD34, and **CD63**), only the percent expression of CD9 was significantly increased on HTLV-I and -II-infected cell lines as compared with that on virus-negative cell lines. Analysis of the beta-1 integrin subfamily (CD29, CD49b, CD49d, CD49e, and CD49f) showed no significant change, except that CD49e was significantly decreased on the HTLV-infected cell lines. For the beta-2 integrin subfamily, the cell surface density was increased for CD18 and CD11a, while the CD11c molecule was expressed exclusively on the HTLV-I- and HTLV-II-infected cell lines. Analysis of several R-CR pairs (CD2-CD58, CD45RO-CD22, CD5-CD72, CD11a-CD54, gp39-CD40, and CD28-CD80) demonstrated that comparable levels of expression of the Rs (CD2, CD45RO, CD5, and CD28) and of some of the CRs (CD58, CD22, and CD72) were in all cell lines; however, CD54, CD40, and CD80 were expressed constitutively on the HTLV-I- and HTLV-II-infected cell lines. Functionally, the expression of these R-CR pairs did not appear to affect the autologous proliferation, since monoclonal antibodies to these R-CR pairs were not able to inhibit proliferation of the infected cell lines. Taken together, our results indicate that HTLV-I ana -II can modulate the expression of several T-cell activation molecules and CRs normally expressed on alternate cell types.

L7 ANSWER 6 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1994:78168 BIOSIS
DOCUMENT NUMBER: PREV199497091168
TITLE: Association of host cell surface adhesion receptors and other membrane proteins with HIV and SIV.
AUTHOR(S): Orentas, Rimas J.; Hildreth, James E. K. (1)
CORPORATE SOURCE: (1) Leukocyte Immunochem. Lab., Johns Hopkins Univ. Sch. Med., Dep. Pharmacol. and Molecular Sci., 725 N. Wolfe St., Baltimore, MD 21205 USA
SOURCE: AIDS Research and Human Retroviruses, (1993) Vol. 9, No. 11, pp. 1157-1165.
ISSN: 0889-2229.
DOCUMENT TYPE: Article
LANGUAGE: English
AB We have developed a MAb-based capture assay to study the association of host cell membrane proteins with HIV and SIV. Class I and II MHC proteins were found to be associated with HIV as previously described. In addition to these molecules a number of other host molecules were found to be acquired by HIV, including CD71, **CD63**, CD43, and CD8. We have demonstrated that the major leukocyte adhesion receptors LFA-1 (CD11A/CD18) and CD44 are also associated with HIV. The level of surface expression of host membrane proteins did not predict relative expression (capture efficiency) of the virus. The use of virus-susceptible indicator cells showed that the assay involved host membrane protein-mediated capture of infectious HIV and SIV particles. Our data indicate that HIV and SIV acquire a number of host membrane proteins including adhesion receptors and that this process may be nonrandom. The acquisition of host cell adhesion receptors by HIV and SIV could have profound effects on the biology of the viruses, including binding, infectivity, and tropism.

L7 ANSWER 7 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1993:171318 BIOSIS
DOCUMENT NUMBER: PREV199395092368
TITLE: Host cell membrane proteins on human immunodeficiency virus type 1 after in vitro infection of H9 cells and blood mononuclear cells: An immuno-electron microscopic study.
AUTHOR(S): Meerloo, Timo (1); Sheikh, Mubasher A. (1); Bloem, Andries C.; De Ronde, Anthony; Schutten, Martin; Van Els, Cecile A. C.; Roholl, Paul J. M.; Joling, Piet (1); Goudsmit, Jaap; Schuurman, Henk-Jan

CORPORATE SOURCE: (1) Div. Histochem. Electron Microscopy, Dep. Pathol. Internal Med., University Hospital, PO Box 85.500, 3508 GA Utrecht Netherlands Antilles
SOURCE: Journal of General Virology, (1993) Vol. 74, No. 1, pp. 129-135.
ISSN: 0022-1317.
DOCUMENT TYPE: Article
LANGUAGE: English

AB Human immunodeficiency virus type 1 (HIV-1)-infected H9 and blood mononuclear cells (MNCs) were studied by immunogold electron microscopy for the presence of HIV-1 gag p24 protein, env gp41 and gp120 proteins, and host cell molecules CD4, CD11a, CD25, CD54, **CD63**, HLA class I and HLA-DR. Uninfected H9 cells and MNC membranes labelled for CD4, HLA class I and class II, and, at low density, CD11a and CD54; lysosomal structures in the cytoplasm labelled for **CD63**. The infected cell surface showed immunolabelling for HIV-1 proteins, as did budding particle-like structures. Immunogold labelling of the cell membrane for CD4 was almost non-existent. The level of immunolabelling for CD11a and CD54 on infected cells was greater than that on uninfected cells; this is presumably related to a state of activation during virus synthesis. Budding particle-like structures and free virions in the intercellular space were immunogold-labelled for all host cell markers investigated. This was confirmed by double immunogold labelling using combination of HIV-1 gag p24 labelling and labelling for the respective host cell molecule. We conclude that virions generated in HIV-1-infected cells concentrate host-derived molecules on their envelope. Also molecules with a prime function in cellular adhesion concentrate on the virion.

L7 ANSWER 8 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1993:34898 BIOSIS
DOCUMENT NUMBER: PREV199395023098
TITLE: Modulation of cell surface molecules during HIV-1 infection of H9 cells: An immunoelectron microscopic study.
AUTHOR(S): Meerloo, Timo; Parmentier, Henk K.; Osterhaus, Albert D. M. E.; Goudsmit, Jaap; Schuurman, Henk-Jan (1)
CORPORATE SOURCE: (1) Div. Histochemistry, Electron Microscopy, Dep. Pathology, Univ. Hosp., P.O. Box 85.500, 3508 GA Utrecht Netherlands Antilles
SOURCE: AIDS (Philadelphia), (1992) Vol. 6, No. 10, pp. 1105-1116.
ISSN: 0269-9370.
DOCUMENT TYPE: Article
LANGUAGE: English

AB Objective: To study cell surface molecules and HIV-1 proteins on H9 cells 2 days after infection by immunogold electron microscopy, either in single or in double labelling using combinations of host cell-derived molecules and HIV-1 proteins. Design and methods: The presence of host cell antigens CD3, CD4 and human leukocyte antigen-DR (HLA-DR) and HIV-1 antigens gag p15, p17, p24 and env gp41 was evaluated using immunocytochemistry at the light microscopic level. H9 cells 2 days after infection were processed for conventional transmission electron microscopy and cryo-ultramicrotomy. Leukocyte antigens investigated were CD2, CD3, CD4 (two antibodies), CD5, CD8, CD25, CD30, **CD63** antigens and HLA-DR; HIV-1-encoded antigens were gag p24, pol reverse transcriptase, and env gp41 and gp120. Double immunogold labelling was performed using reagents with different sized gold particles. For leukocyte markers, the labelling density of the cell membrane was assessed quantitatively on uninfected and infected H9 cells. Results: Infected cells revealed the presence of gag p24, pol, and env gp41 and gp120 antigens on HIV-1 virions. Uninfected H9 cells showed a random distribution of cell surface molecules, including CD4 antigen, along the plasma membrane. The **CD63** antigen, a lysosomal membrane glycoprotein, was located mainly in the cytoplasm of uninfected cell. Cells 2 days after infection showed CD4 labelling on sites where

virions were budding from or attached to the cell surface and on free virions. Virions also showed labelling by CD3, CD5, CD25, CD30 and **CD63** antibodies and anti-HLA-DR. Compared with uninfected cells, a significantly lower density was found on infected cells in labeling for CD4, CD5, and anti-HLA-DR. A significantly higher density on cells 2 days after infection was seen in **CD63** labelling. Conclusions: During the first phase of infection host cell molecules concentrate on budding structures and newly generated HIV-1 virions. This phenomenon might contribute to the disappearance of these molecules (like the CD4 molecule) from the cell membrane and infection.

L7 ANSWER 9 OF 10 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1993:24612 BIOSIS
DOCUMENT NUMBER: PREV199395012812
TITLE: C33 antigen recognized by monoclonal antibodies inhibitory to human T cell leukemia virus type 1-induced syncytium formation is a member of a new family of transmembrane proteins including CD9, CD37, CD53, and **CD63**.
AUTHOR(S): Imai, Toshio; Fukudome, Kenji; Takagi, Shin; Nagira, Morio; Furuse, Mikio; Fukuhara, Norio; Nishimura, Miyuki; Hinuma, Yorio; Yoshie, Osamu
CORPORATE SOURCE: Shionogi Inst. Med. Res., 2-5-1 Mishima, Settsu-shi, Osaka 566 Japan
SOURCE: Journal of Immunology, (1992) Vol. 149, No. 9, pp. 2879-2886.
ISSN: 0022-1767.
DOCUMENT TYPE: Article
LANGUAGE: English
AB C33 Ag was originally identified by mAb inhibitory to syncytium formation induced by human T cell leukemia virus type 1. The Ag was shown to be a highly heterogeneous glycoprotein consisting of a 28-kDa protein and N-linked oligosaccharides ranging from 10 to 50 kDa. In the present study, cDNA clones were isolated from a human T cell cDNA expression library in Escherichia coli by using mAb C33. The identity of cDNA was verified by immunostaining and immunoprecipitation of transfected NIH3T3 cells with mAb. The cDNA contained an open reading frame of a 267-amino acid sequence which was a type III integral membrane protein of 29.6 kDa with four putative transmembrane domains and three putative N-glycosylation sites. The C33 gene was found to belong to a newly defined family of genes for membrane proteins, such as CD9, CD37, CD53, **CD63**, and TAPA-1, and was identical to R2, a cDNA recently isolated because of its strong up-regulation after T cell activation. Availability of mAb for C33 Ag enabled us to define its distribution in human leukocytes. C33 Ag was expressed in CD4+ granulocytes. Its expression was low in CD8+ T cells and mostly negative in CD16+ NK cells. PHA stimulation enhanced the expression of C33 Ag in CD4+ T cells by about 5-fold and in CD8+ T cells by about 20-fold. PHA stimulation also induced the dramatic size changes in the N-linked sugars previously shown to accompany human T cell leukemia virus type 1-induced transformation of CD4+ T cells.

L7 ANSWER 10 OF 10 CA COPYRIGHT 2002 ACS
ACCESSION NUMBER: 127:80164 CA
TITLE: Single-chain antibodies with membrane-binding domains that mediate adhesion between cells and their use as co-stimulatory ligands
INVENTOR(S): Ledbetter, Jeffrey A.; Hayden, Martha; Fell, Perry; Mittler, Robert; Winberg, Gosta
PATENT ASSIGNEE(S): Bristol-Myers Squibb Company, USA
SOURCE: PCT Int. Appl., 69 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9720048	A2	19970605	WO 1996-US19051	19961127 <--

W: CA, JP, MX
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1995-7755P P 19951130
AB Single-chain antibodies (sFv mols.) with membrane-binding domains are described. These sFv mols. stimulate adhesion between CD4+ T-cells and antigen-presenting cells thereby increasing the immune response against disease. The antigen binding domain binds a leukocyte antigen and transmembrane domain is derived from a cell surface receptor, specifically a leukocyte antigen. **Retrovirus** expression vectors for sFv's using monoclonal antibodies to neural cell adhesion mol. L1 with the transmembrane domain of B7 or CD58 were constructed by std. methods. Expression of the constructs in animal cell lines led to surface presentation of the antibody.

=> d his

(FILE 'HOME' ENTERED AT 14:58:10 ON 30 JUL 2002)

FILE 'BIOSIS, MEDLINE, SCISEARCH, CA' ENTERED AT 14:59:28 ON 30 JUL 2002
L1 1690 S CD63
L2 59597 S LENTIVIR?
L3 0 S L1 AND L2
L4 257762 S RETROVIR?
L5 12 S L1 AND L4
L6 11 DUP REM L5 (1 DUPLICATE REMOVED)
L7 10 S L6 AND PY<=2000

=> s l1 with l4
MISSING OPERATOR L1 WITH
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l1 (w) l4
L8 0 L1 (W) L4

=> s l1 (s) l4
L9 0 L1 (S) L4

=> d his

(FILE 'HOME' ENTERED AT 14:58:10 ON 30 JUL 2002)

FILE 'BIOSIS, MEDLINE, SCISEARCH, CA' ENTERED AT 14:59:28 ON 30 JUL 2002
L1 1690 S CD63
L2 59597 S LENTIVIR?
L3 0 S L1 AND L2
L4 257762 S RETROVIR?
L5 12 S L1 AND L4
L6 11 DUP REM L5 (1 DUPLICATE REMOVED)
L7 10 S L6 AND PY<=2000
L8 0 S L1 (W) L4
L9 0 S L1 (S) L4

=> s l1 (w) (inhib or reduc?)
L10 0 L1 (W) (INHIB OR REDUC?)

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=> s cd63 (w) (inhib? or reduc?)
L11           1 CD63 (W) (INHIB? OR REDUC?)

=> d l11 ibib abs

L11  ANSWER 1 OF 1  BIOSIS  COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER:   2001:168303  BIOSIS
DOCUMENT NUMBER:    PREV200100168303
TITLE:              Profound inhibition of GPIb, GPIIb/IIIa, PECAM-1, CD63, and
                    CD107 in a chronic drug addict: Selecting controls for
                    platelet flow cytometry in the inner city hospital.
AUTHOR(S):          Bell, Christopher R.; Horowitz, Eric D.; Oshrine, Benjamin
                    R.; Serebruany, Victor L. (1)
CORPORATE SOURCE:   (1) Center for Thrombosis Research, Sinai Hospital of
                    Baltimore, 2401 West Belvedere Avenue, Schapiro Research
                    Building R 202, Baltimore, MD, 21215: heartdrug@aol.com USA
SOURCE:             Thrombosis Research, (February 1, 2001) Vol. 101, No. 3,
                    pp. 217-218. print.
                    ISSN: 0049-3848.
DOCUMENT TYPE:      Article; Letter
LANGUAGE:           English
SUMMARY LANGUAGE:   English
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